

~~CONFIDENTIAL~~

~~CONFIDENTIAL~~

DATE OF INFORMATION 1949

DATE DIST. 15 Dec 1949

NO. OF PAGES 5 50X1-HUM

SUPPLEMENT TO  
REPORT NO. 50X1-HUM

50X1-HUM

THIS IS UNEVALUATED INFORMATION

MORPHOLOGIC CHARACTERISTICS OF ACUTE ATTACKS  
IN CHRONIC BRUCELLOSIS

Chronic brucellosis is quite frequently observed in endemic centers -- from 45 to 75 percent of the cases (Ragoza). According to our data, 60 percent of the cases are chronic brucellosis.

As we know, symptoms of chronic brucellosis are detected after the onset of acute and subacute stages. In general, the acute, subacute, and chronic stages indicate protean manifestations. Exacerbation of chronic brucellosis and transition of the pathogenic process to an acute stage are also possible. The chief symptoms of chronic brucellosis are its protracted course, clinically asymptomatic, frequent negative agglutination reaction, and extensive dystrophic and inflammatory processes in the organs and tissues resulting in sclerosis. The tendency of the inflammatory process toward frequent exacerbation is typical. As shown by morphological studies, definite sclerotic changes appear 6 months after the onset of the disease. Based on the above findings, it can be assumed that this evolution of acute brucellosis into the chronic form takes place during this period.

It is extremely difficult to diagnose chronic brucellosis because the clinical symptoms are uncertain and the Wright reaction is variable. Since the Wright reaction is often negative, clinical data remain the basis of diagnosis.

**CONFIDENTIAL**

- 1 -

~~CONFIDENTIAL~~

Sanitized Copy Approved for Release 2011/09/19 : CIA-RDP80-00809A000600270278-4

50X1-HUM

CONFIDENTIAL

CONFIDENTIAL

In the chronic stage, either a slow fading out of protective reactions with an increase in general intoxication sets in or there is an increase in general resistance with a lowering of local resistance in individual organs. This condition is a prerequisite for localized infection.

We obtained 26 sections from cases 40 to 60 years old, who died of brucellosis after 6 months to 3 years.

The following forms of brucellosis were observed: 1) nonreactive or cachectic; 2) immunoreactive or visceral.

The nonreactive (cachectic) form of brucellosis is illustrated by the following example; Patient K., 45 years old; died after a one-month stay in the hospital. Chronic brucellosis, brucellar meningitis and central pneumonia were clinically established. The patient, a veterinary worker, was in frequent contact with animals with brucellosis and included unprocessed dairy products in his diet. He contracted acute brucellosis 9 months earlier with fever and pain in the joints. After a short remission, the disease took a chronic course. A subfebrile temperature was noted with pains in the small of the back and the large joints, which confined the patient to bed. An enlargement of the liver, hypochromic anemia, and leukopenia with relative lymphocytosis were observed. The sedimentation rate was accelerated. The cerebrospinal fluid specimen indicated a positive Nonne-Apelte and Pandy reaction and 3 percent of albumin. The section showed a definite general exhaustion with atrophy of the parenchyma and sclerosis of the stroma of the internal organs. The cause of death was determined as serous meningitis and pneumonia which developed in the final stage of the disease.

The nonreactive (cachectic) form of chronic brucellosis is characterized by its protracted course. The clinical symptoms indicated lingering fever, pains in the bones and joints, enlargement of the liver and spleen, progressive anemia and cachexia. Cachexia can occur with or without edema. With this background intercurrent diseases are apt to develop: central pneumonia, sepsis and tuberculosis. Histologically, marked dystrophias of the organic parenchyma and vascular and endocrine systems, with round cell, inflammatory infiltration of interstitial laminae, and subsequent sclerosis are observed.

Immunoreactive attacks of chronic brucellosis occur in the form of recurrent septic and visceral brucellosis.

Recurrent septic brucellosis is illustrated by the following case:

Patient L., 20 years old, official of the War Office, became ill with brucellosis a year ago. Fever, general malaise and a positive Wright reaction in 1:200 were noted. Later the brucellosis ran a chronic course with periodic exacerbations. On admission to the hospital, patient complained of a high fever, which had developed 3 days previously, sudoresis, headache, pains in the joints, and asthenia. Clinical symptoms of high fever, weak, rapid pulse, dull heart tone, tachycardia, shortness of breath, and enlargement of the liver and spleen were observed. Some months after hospitalization, patient died from increased cyanosis and heart failure.

Blood analysis on 7 February 1945: Hb 65 percent; erythrocytes 3,860 [sic]; color index 0.8; leukocytes, 2,100; "p." 3; s, 17; lymphocytes 77; sedimentation rate, 30 millimeters per hour.

Clinical diagnosis: exacerbation of chronic brucellosis.

The cause of death was determined as a collapse due to infectious toxic origin.

- 2 -

CONFIDENTIAL

CONFIDENTIAL

50X1-HUM

CONFIDENTIAL

Histologically, diffused interstitial (histiocytic) myocarditis and hepatitis, catarrh, hemorrhagic lymphadenitis, and infectious splenitis with hyperplasmia in the reticulo-endothelial system were indicated.

Hence, recurrent septic brucellosis is a flare-up of a chronic process. This type of infection is manifested by high temperature of the undulant or sporadic form, sudden enlargement of the liver and spleen, disruption of the heart, blood vessels, motor apparatus, and vegetative endocrine system. Morphologically brucellosis indicates a combined form of histiocytic myocarditis and hepatitis, and also of progressive, hemorrhagic lymphadenitis and splenitis. Inflammatory changes in the nidi may also be observed in the suprarenal and vegetative nerve ganglia, which, combined with the disruption in the cardiovascular system, can cause death due to collapse.

The clear manifestation of the clinical symptoms of recurrent septic brucellosis and the peculiarity of the morphological reactions (histiocytic proliferation and hemorrhagic edema) indicated the importance of allergies in these cases.

Visceral brucellosis is characterized by the prevalence of organic diseases of the central nervous, hepato-lienal, and cardiovascular systems. Affection of the central nervous system is most frequently observed in the chronic stage. The symptoms are manifested by neurasthenia or psychasthenia (Serafimov) or may resemble meningitis, meningo-encephalitis and, less frequently, myelitis (Ochkur, Roger and Pourcines). Neurological symptoms develop against a background of general brucellosis infection.

Histologically meningitis is a diffused, sero-lympho-histiocytic disease. The pia mater is swollen, dull and of a whitish color. Greyish ganglia, the size of a pin head, and isolated subarachnoid hemorrhages are found in it. The inflammatory process usually begins in the pia mater and subsequently spreads into the cerebral tissue. Encephalitic brucellosis may be centralized (more frequently) or diffused. Under the microscope, ganglia composed of microglia with a considerable number of histiocytes are found in the cerebral cortex, its white matter, and in the subcortical ganglia.

Myelitis is accompanied by degeneration of nerve cells in the gray matter of the spinal cord, decay of the myelin and nerve fibers, and also by the development of centers of perivascular, round-cell, inflammatory infiltration. Its course is accentuated in the region of the cervical and lumbar processes and leads to a lateral disturbance of the spinal cord with subsequent paralysis of the limbs and rectal and vesical sphincters.

Hepato-lienal syndrome is well defined in the majority of chronic brucellosis cases. The enlargement of the liver and spleen, ascites, jaundice, and exhaustion can be clinically determined; hemorrhagic diathesis can also be noted. Histologically, the hepatopathy presents symptoms of mixed cirrhosis. The onset of the disease is marked by serous inflammation of the liver and multiplication of Kupffer's cells. Progressive inflammation follows. Dystrophias and degeneration of hepatic parenchyma occur simultaneously. The degenerated parts of the hepatic lobules are replaced by new granular tissues composed of epithelioid, histiocytic, and lymphoid cells. In the final stage of cirrhosis, the atrophied hepatic lobes are surrounded by streaks of fibrous connective tissue, infiltrated lymphocytes, and plasmocytes.

Hyperplasia of the cells of the reticulo-endothelial system may be seen in the spleen. Collagenic changes take place in the reticular stroma. Sidero-fibrous ganglia may appear in the replacement of degenerated tissue indicating connective tissue impregnated with calcium and iron. Due to fibrous degeneration the lymph follicles of the spleen decrease in number and volume.

- 3 -

CONFIDENTIAL

CONFIDENTIAL

50X1-HUM

The course of cardiac brucellosis is the same as that of chronic bacterial endocarditis combined with interstitial and, more rarely, fibro-hemorrhagic pericarditis. The following case is cited as an example of pericardial brucellosis.

Patient K, 59 years of age, had suffered from brucellosis for 11 months. The disease began with an acute attack and a high temperature in August 1945. On admission, he complained of vertigo, pains in the cardiac region, shortness of breath, and swellings in the lower half of the body. It was found that he had had recurrent angina and had been a typhoid carrier in the past.

Clinical symptoms were manifested by systolic sound in the upper region of the aorta and diastolic sound under it; heart sounds were dull and the heart was dilated. Swelling of the lower limbs and ascites were present.

Blood analysis disclosed acute anemia (Hb 33 percent; erythrocytes 1,360,000) and neutrophil leukocytosis.

Death was due to increased weakness of the heart action.

A pathological diagnosis revealed chronic brucellosis; Brucellar pancarditis, polypous, ulcerative endocarditis of the aortic valve; interstitial myocarditis and fibrohemorrhagic pericarditis; chronic hyperplasia of the spleen; congested liver, ascites, anasarca; exacerbation of bilateral, and apical tuberculosis.

An inflammatory infiltration of histiocytes with an admixture of eosinophils and plasmocytes was noted in the tissue of the aortic valve. The superficial layers of the valve had become necrotic with a considerable deposit of fibrin and petrification centers. Against a background of reticular sclerosis, a centralized, histiocytic proliferation around the vessels was observed in the myocardium. Micronecrosis centers were found in the pericardium as well as moderate round-cell and histiocytic inflammatory infiltrations.

Heart disease in cases of brucellosis is frequently the cause of death. As a rule, it appears in the form of subacute or chronic endocarditis affecting the aortic and mitral valves and develops into the subacute or chronic stage of brucellosis. It is frequently combined with disease of the central nervous system as the result of embolisms and extensive vasculitis. Chronic brucellosis can aggravate healed tubercular centers because of the disappearance of general and local immunity. A case of brucellosis complicated by tuberculosis will be reported separately at a later date.

#### CONCLUSIONS

1. Brucellosis generally runs a cyclic course with protean manifestations of acute, subacute, and chronic stages depending upon the immunobiological changes in the individual. In addition, it is possible to find cases indicating symptoms of chronic brucellosis for the first time.

2. Chronic brucellosis is very prevalent in endemic centers. Its progress depends upon the general and local resistance of the case to the development of exacerbation, general intoxication, and cachexia or against organic diseases which can bring about death.

CONFIDENTIAL

- 4 -

CONFIDENTIAL

50X1-HUM

CONFIDENTIAL **CONFIDENT**

BIBLIOGRAPHY

- Ariel', M. B., "Pathologic Morphology of Brucellosis," 1939; Arkh. Biol. Nauk., 5, pp 2-3, 1940
- Zhukhin, V. A. "Pathologic Anatomy of Brucellosis in Man," Ufa, 1942
- Myasnikov, "Brucellosis Clinic," L, 1944
- Novitskiy, "Pathologic Anatomy of Brucellosis in Man," Tr. Omskogo Med. In-ta, 8, 1914
- Ochkar, Biell. Eksp. Biol i Med., 3, 1945; ibid, 3, 1946; Arkh. Patol., 8, pp 1-2, 1946; Izv. Akad Nauk. Est. SSR, 3, 1946
- Ragoza, Klin. Med. 2, 1941
- Serafimov, Nevropatol. i Psikhiatr., 7, 1937
- Steblov, Nevropatol. i Psikhiatr., 7, 1939
- Ulintsev, Izv. Akad Nauk Est. SSR, 31, 1946
- Ghalisov, Arkh. Patol. Anatom. i Patol. Fiziol., 1, 1939 and 1941
- Shlyapnikov, Tr. Gesp. Privezhsk. Vozen. Okruga, 1944
- Roger et Pourcines, "Meningo-neurobrucellosis," Paris, 1938

- E N D -

**CONFIDENTIAL**

- 5 -

CONFIDENTIAL